

A CLINICAL AND EXPERIMENTAL STUDY OF THE ELECTROCARDIOGRAPHIC CHANGES IN EXTREME ACIDOSIS AND CARDIAC ARREST

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This investigation of the electrocardiographic and biochemical changes in very severe acidosis was prompted by the study of a patient with cardiac arrest (Stewart, Stewart, and Gillies, 1962) in whom improvement of myocardial activity appeared to be correlated with correction of acidosis. This paper describes clinical and experimental data indicating that heart block and asystole may be caused by metabolic acidosis and supporting the view (Stewart, 1964) that acidosis may sometimes prevent restoration of cardiac function. Acid-base measurements and electrocardiograms were obtained when practicable in clinical cases of cardiac arrest and in the experimental animals metabolic acidosis was induced by infusion of 0.3 M HCl. We believe that these studies have important practical applications in the clinical management of patients especially where electrocardiographic monitoring is used in conjunction with external cardiac massage.

CLINICAL DATA

Case 1 (W.S., aged 1 year). This patient had a gangrenous appendix removed and was being treated after operation with intravenous fluids, antibiotics, and gastric suction. Twelve hours after the operation he became severely shocked due to inhalation of vomitus. In spite of continued suction, inhalation occurred a second time and on this occasion cardiac arrest followed. Thoracotomy was immediately carried out and internal massage commenced. The heart was in asystole but spontaneous beats returned 40 minutes later. At first they were of poor quality with weak peripheral pulses and some improvement followed the administration of intracardiac adrenaline. Thereafter, however, the quality of the heart beat deteriorated, at first with four-to-one block and later with no ventricular activity. No response was now obtained to stimulation with adrenaline or to administration of calcium salts, and further attempts at resuscitation were abandoned. The patient was treated before the importance of correcting acidosis was known.

Case 2 (W.L., aged 4 months). Cardiac arrest occurred during angiocardigraphic investigation of cyanotic heart disease. There had been no untoward reaction to a test dose of contrast medium, but the main injection was immediately followed by ventricular fibrillation. The chest was opened after four minutes of external cardiac massage and defibrillation was achieved by electrical counter-shock. At first the heart beat was rather feeble and the electrocardiogram showed complete heart block. After internal massage had been continued for 60 minutes the heart was able to maintain a satisfactory circulation and the chest was closed. The patient was transferred to the ward but died 30 minutes later in peripheral circulatory failure. A venous blood sample taken 15 minutes before death revealed a pH of 6.67. At necropsy mitral atresia with fibro-elastosis of the endocardium of the left atrium, transposition of the great vessels, and a displaced ventricular septum with a large ventricular septal defect were found.

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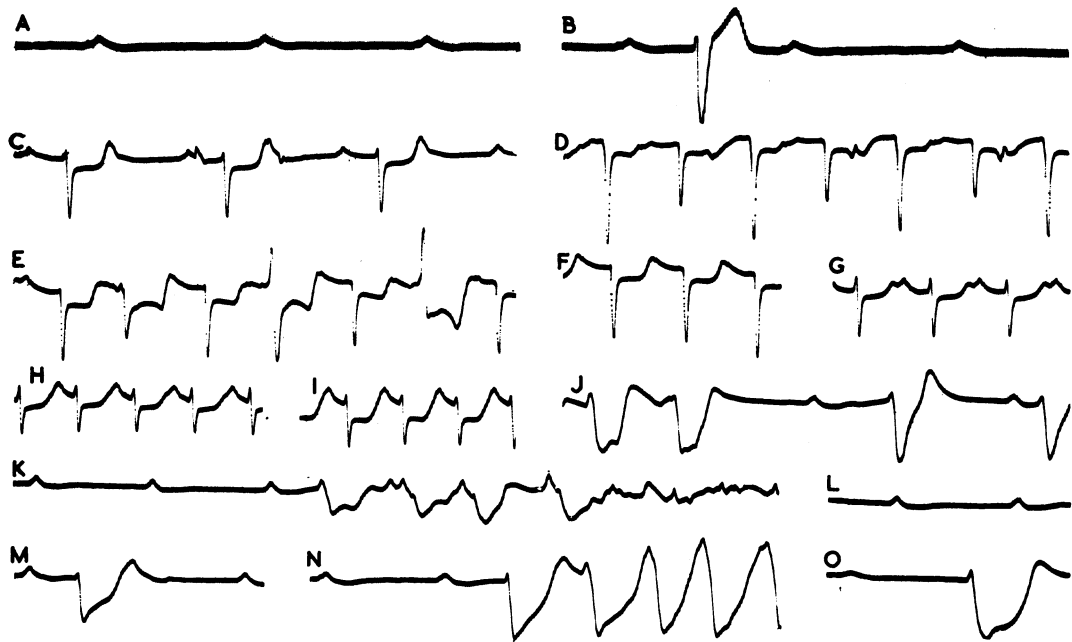


FIG. 1—Illustrating *clinical results* (electrocardiograms from patient, Case 3; all records are lead II). Ventricular asystole (A) and complete heart block (B) after 20 minutes of cardiac massage and lung inflation. Two-to-one heart block with prolonged P-R interval and S-T depression in the conducted beats after 70 minutes of massage and vigorous inflation of the lungs (C). Electrical alternans shortly after the bicarbonate infusion was started (D). This and the subsequent two traces (E, F) are virtually continuous. Sinus rhythm with prolonged P-R interval, more prominent S-T depression, T inversion, and coupled ventricular ectopic beats which vary in form (E). Sinus rhythm with prolonged P-R interval, T waves now upright are superimposed on the P waves, approximately four minutes after bicarbonate (F). Sinus rhythm with P-R prolongation 17 minutes after bicarbonate, the P and T waves intersect (G). Sinus tachycardia, with superimposed P and T waves, at completion of operation, before inflation was stopped (H) and 11 minutes later (I). Irregular ventricular complexes and heart block at 12 minutes (J); blood pressure was unrecordable. Ventricular asystole and ventricular fibrillation at 15 minutes (K) with spontaneous reversion to ventricular asystole at 16 minutes (L). Ventricular asystole was terminated by external massage, and bizarre ventricular complexes were recorded at 17 minutes (M), 22 minutes (N), and 30 minutes (O).

Case 3 (M.T., aged 12 years). A few hours after a head injury this patient was admitted unconscious and with gasping respirations. Just before arrival at the ward he vomited and within a few seconds respiration ceased. External cardiac massage and expired air ventilation were started at once and endotracheal intubation and inflation of the lungs with oxygen were substituted a few minutes later. The pupils were widely but not fully dilated at this time, the fundi showed papilloedema with hæmorrhages, and the tendon reflexes were absent. Approximately 20 minutes after commencing external massage an electrocardiogram showed complete heart block. This changed to ventricular asystole (Fig. 1A) within seconds, but after restarting massage and ventilation complete heart block (Fig. 1B) was restored. The endotracheal tube was changed for a better fitting one and vigorous inflation of the lungs was carried out. An electrocardiogram obtained at this time, 40 minutes after commencing massage, showed two-to-one heart block with prolonged P-R interval and S-T depression in conducted beats. This dysrhythmia persisted (Fig. 1C) until bicarbonate infusion 30 minutes later. The patient was transferred to the operating theatre and the long saphenous vein was cannulated at the groin. After approximately 70 minutes of massage, when the electrocardiogram still showed two-to-one heart block, 150 mEq of sodium bicarbonate solution (2½%) was rapidly infused and there was an immediate improvement in the electrocardiogram, which coincided with the return of palpable peripheral pulses. The exact timing was not recorded on the electrocardiograms but the sequence included two-to-one heart block with increased S-T depression (Fig. 1C), electrical alternans (Fig. 1D), and irregular ventricular complexes (Fig. 1E), before sinus rhythm was sustained (Fig. 1F). The heart block (Fig. 1C) was present before the infusion started and external cardiac massage was discontinued approximately 4

minutes after the start of the infusion when sinus rhythm (Fig. 1F) was present. The venous blood pH was 7·08, peripheral pulses were strong, and the heart was clearly in sinus rhythm with long P-R interval and distinct P and T waves (Fig. 1G) 17 minutes after the bicarbonate infusions. Craniotomy was done and an extradural hæmatoma evacuated. At the end of this operation there was no evidence of spontaneous respiration and it was concluded that coning, with irreversible cerebral damage, had occurred three hours previously, at the time of admission. Further attempts at resuscitation were, therefore, abandoned, but subsequently the effect on the electrocardiogram of recommencing external cardiac massage was observed. The heart was beating strongly and regularly when inflation was stopped (Fig. 1H) and 11 minutes later (Fig. 1I). At 12 minutes it suddenly became irregular (Fig. 1J) and at approximately 15 minutes (Fig. 1K) the electrocardiogram showed, successively, heart block, ventricular asystole, ventricular extrasystoles, and ventricular fibrillation. This last dysrhythmia reverted spontaneously to ventricular asystole (Fig. 1L) which was terminated by external massage. A record during intermission of massage showed complete heart block with idio-ventricular rhythm, and bizarre ventricular complexes were recorded at 17 minutes (Fig. 1M), 22 minutes (Fig. 1N), and 30 minutes (Fig. 1O). The presence of coning was confirmed at necropsy at which were also shown posterior myocardial contusion and inhalation bronchopneumonia.

MATERIAL AND METHODS

Ayrshire bull calves less than 8 days old of weight 30–38 kg. were used in the experiments. General anaesthesia was induced with intravenous sodium thiopentone (0·5–0·75 g.), and at the same time intravenous atropine (1 mg.) and pethidine (100 mg.) were given. Anaesthesia was continued as necessary with small supplementary doses of sodium thiopentone. A cuffed endotracheal tube was passed either transorally or, if difficulty was encountered, through a tracheostomy. Intermittent positive pressure inflation with pure oxygen was maintained with a mechanical respirator. Cannulae for infusions, pressure monitoring, and blood sampling were inserted into the abdominal aorta and inferior vena cava. In all experiments the calf was lying on its right side and there was intermittent monitoring of lead II of the electrocardiogram with a direct writing electrocardiograph, of mean blood pressure with a mercury manometer, and of oesophageal temperature with a thermocouple. Intermittent arterial blood samples were obtained. An intravenous infusion of 0·3 M. hydrochloric acid, given at an approximate rate of 10–20 ml./min., was continued, except for blood sampling, until cardiac arrest occurred. An infusion of 100 ml. of 0·3 M. sodium bicarbonate was then given as rapidly as possible, usually in 20–40 seconds, while the circulation was maintained with external cardiac massage. Since the aim of the experiment was to simulate clinical conditions no special attempt was made to prevent the development of respiratory acidosis, hyperkalaemia, and anoxia, which occurred under the experimental conditions and are also common following cardiac arrest in clinical practice.

RESULTS

After induction of anaesthesia standard lead electrocardiograms were obtained (Fig. 2 A–D). Infusion of acid caused hypertension which subsided whenever the infusion was stopped, as was done for about one to two minutes before each blood sampling to allow for some circulatory mixing. When the results were analysed it was clear that there was a consistent pattern in 6 of the 10 animals.

Typical Electrocardiographic Changes. With severe acidosis there was tachycardia with a deep QS wave (Fig. 2F). With very severe acidosis some spontaneous respiratory efforts occurred, and the electrocardiogram showed a high T and a very deep S (Fig. 2F). In extreme acidosis the usual electrocardiographic sequence was: electrical alternans (Fig. 2G); bradycardia with two-to-one heart block (Fig. 2H); complete heart block (Fig. 2J) and ventricular asystole (Fig. 2K). In two animals a complex rhythm (Fig. 2I) was seen, usually during rapid acid infusion, and in one animal (Calf 8), in which two-to-one heart block was not seen, the P waves were not regular in extreme acidosis and ventricular asystole was preceded by variable heart block. In all six animals the blood pressure, which fell rapidly from the time of onset of bradycardia, was maintained by external cardiac massage and a normal blood pressure was restored in less than five minutes after bicarbonate infusion. In all six animals with the usual sequence during induction of acidosis, the electrocardiograms taken during intermissions of massage and after bicarbonate infusion showed progressive improvement with reversal of the sequence noted above: ventricular asystole, complete heart block, two-to-one

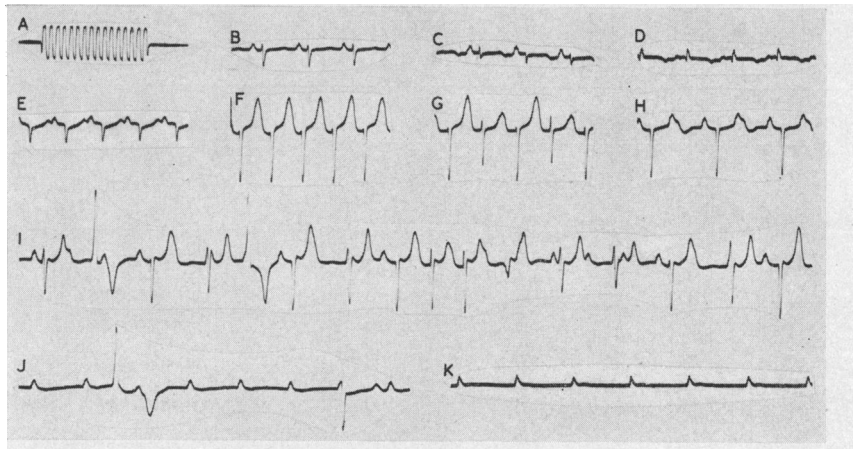


FIG. 2.—Illustrating *typical experimental results*. Standardization (A) and standard leads I (B), II (C), and III (D). All subsequent traces (E to K) are lead II. They show the progressive effect of acid infusion. Sinus tachycardia (E), deep S and high T with superimposed P and T (F), electrical alternans (G), and two-to-one heart block (H). Complex rhythm with atrio-ventricular dissociation and interference (I). Complete heart block with gross bradycardia and varying idio-ventricular centre in extreme acidosis (J). Ventricular asystole (K).

heart block, electrical alternans, and sinus tachycardia. In the course of these changes there was usually only a minimal rise in the plasma potassium level and a slight fall in temperature, usually about two centigrade degrees.

Atypical Electrocardiographic Changes. Different and variable electrocardiographic results (Fig. 3) were obtained in four animals. The heart appeared unusually irritable in Calf 2 (Fig. 3A) and hypotension with bradycardia and broad QRS complexes soon supervened (Fig. 3B). In

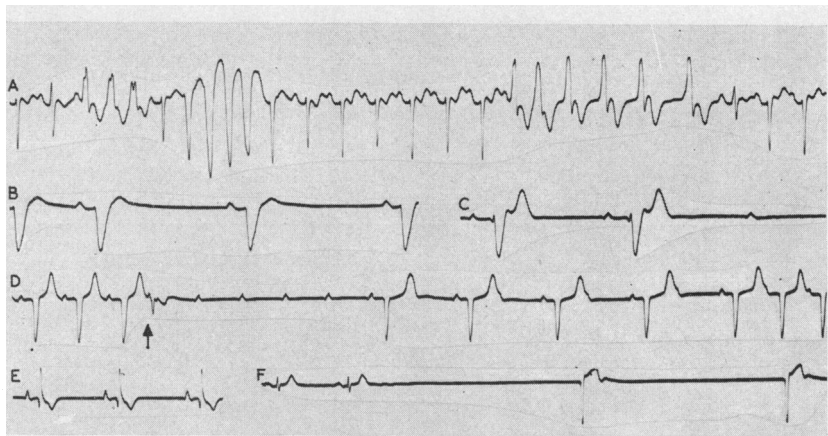


FIG. 3.—Illustrating *atypical experimental results* (all records are lead II). Irritable heart in Calf 2 showing successively: multifocal ventricular ectopic beats, a short burst of ventricular tachycardia, electrical alternans, ventricular tachycardia and sinus tachycardia (A). Broad RS complexes with increasing bradycardia from Calf 2 (B). Bradycardia with broad RS complexes changing to ventricular asystole, from Calf 10 (C). Hypotension was associated in both instances. During severe acidosis in Calf 10 præcordial percussion, indicated by the arrow, is followed by temporary ventricular asystole and two-to-one heart block with reversion to normal rhythm (D). Bradycardia with relatively normal electrocardiogram in Calf 5 (E). Sinus rhythm with RS-type initial ventricular pattern followed by sinus arrest and an idioventricular rhythm which originates in or near the atrio-ventricular node and shows retrograde conduction into the atrium in Calf 6 (F).

TABLE I
DETAILED EXPERIMENTAL DATA FROM CALF 3

Specimen	Time (min.)	Biochemical data (arterial blood)					Mean B.P. (mm. Hg)	Electrocardiogram	Change since last specimen
		pH	CO ₂ content (mMol./l.)	PCO ₂ (mm. Hg)	K (mEq/l.)	O ₂ (% sat.)			
1	0	7.28	27	55	4.5	97	80	Control	—
2	35	7.05	23	78	4.1	98	90	Sinus tachycardia	+75 mEq HCl
3	68	7.10	18	55	3.9	98	110	Sinus tachycardia	+75 mEq HCl
4	75	6.95	23	94	3.9	42	150	Electrical alternans	Stop inflation 7 min.
5	79	7.06	21	69	3.8	98	110	Sinus tachycardia	Re-inflation 4 min.
6	83	7.10	21	64	4.0	100	110	Sinus tachycardia	Inflation 4 min.
7	103	7.03	18	63	3.8	96	115	Sinus tachycardia	+60 mEq HCl
8	152	6.96	14	56	4.1	95	125	Electrical alternans	+45 mEq HCl
9	179	6.70	9	60	4.8	95	145	2:1 heart block	+45 mEq HCl
10	193	6.55	6	53	5.3	96	160	Irritable heart	+75 mEq HCl
11	205	6.09	2	c33	5.9	90	50	Complete heart block	+15 mEq HCl
12	206	6.32	2	c25	5.9	84	60	Complete heart block	Wait 1 min.
13	210	6.81	12	66	5.3	96	120	2:1 heart block	+60 mEq NaHCO ₃
14	212	6.72	10	65	5.3	96	160	Sinus tachycardia	Wait 2 min.
15	224	6.72	9	59	5.4	94	160	Sinus tachycardia	Wait 12 min.
16	240	6.45	11	113	6.6	5	0	Dying heart	Stop inflation 16 min.

c = approximately.

TABLE II
EXPERIMENTAL DATA

Calf No.	Weight (kg.)	Total acid		Time to arrest (min.)	Biochemical results (arterial blood) before arrest			Presence or absence of typical electrocardiographic sequence
		(ml.)	(mEq/kg.)		pH	PCO ₂ (mm. Hg)	K (mEq/l.)	
<i>Typical</i>								
1	30	550	5.5	60	6.59	99	5.0	+
3	35	1300	11.1	204	6.09	60	3.8	+
4	38	750	6.9	50	—	—	—	+
7	33	1250	11.4	70	5.98	c50	6.1	+
8	31	700	6.9	45	6.91	86	4.4	+
9	32	800	5.6	38	6.77	71	4.6*	+
<i>Atypical</i>								
2	32	650	6.1	55	6.78	92	8.6	—
5	31	200	1.9	5	6.66	120	5.8*	—
6	34	600	5.3	33	6.59	114	7.8*	—
10	36	1500	12.5	186	5.42	low	7.0	—

* Lysis present.

calf 10 also, there was hypotension, bradycardia, and broad QRS complexes (Fig. 3C). This animal was very resistant to acidosis and though after some acid (8 mEq/kg.) præcordial percussion was followed by temporary ventricular asystole and two-to-one heart block (Fig. 3D) arrest did not occur until more acid (12.5 mEq/kg.) had been given. In Calf 5 there was hypotension and bradycardia with inconspicuous electrocardiographic changes (Fig. 3E) after relatively little acid (1.9 mEq/kg.). Tachycardia and electrical alternans occurred very early in this animal and ventricular fibrillation developed during external cardiac massage. This was the only animal that showed ventricular fibrillation. A retroperitoneal hæmatoma was found at necropsy. In Calf 6 there was hypotension and bradycardia with a change from sinus rhythm with RS pattern to idio-ventricular rhythm (Fig. 3F).

Detailed Data. Detailed data from a typical experiment are given (Table I) and the results for all 10 animals are summarized in respect of biochemical (Table II) and electrocardiographic (Table III) data. It was first noticed when changing an oxygen cylinder, and subsequently in several animals, that tracheal occlusion, or absence of inflation in animals not breathing spontaneously, was associated with development of the arrhythmia expected next in the typical sequence. During the experiment detailed (Table I) acid infusion was stopped and tracheal occlusion during sinus rhythm was followed by electrical alternans which reverted to sinus rhythm on re-inflation. The pH level at the time of electrical alternans was closely similar to that at which electrical alternans occurred during the subsequent acid infusion.

TABLE III
EXPERIMENTAL ELECTROCARDIOGRAPHIC DATA

Experimental conditions	Dysrhythmia	Typical						Atypical			
		1	3	4	7	8	9	2	5	6	10
Acid infusion	Sinus tachycardia	+	+	+	+	+	+	+	+	+	+
	Electrical alternans	+	+	+	+	+	+	+	+	+	+
	2:1 heart block	+	+		+		+		+		+
	Complete heart block	+	+				+				+
External cardiac massage	Ventricular asystole			+	+	+	+				
Bicarbonate infusion . .	Complete heart block	+	+	+	+	+	+				
	2:1 heart block	+	+	+	+		+				
	Electrical alternans	+		+		+	+				
	Sinus tachycardia	+	+	+	+	+	+				

DISCUSSION

The present electrocardiographic results in the intact bull calf are similar to those in the isolated perfused dog heart (Andrus and Carter, 1924) but different from those reported in the intact dog in which there was said to be no heart block or asystole during acidosis of metabolic (Gertler, Hoff, and Humm, 1946) or respiratory (Brown and Miller, 1952) type. The discrepancies might well be due to differences in technique. For example, artificial ventilation was not used in the metabolic acidosis experiments of Gertler *et al.* (1946). A species difference is less likely because similar arrhythmias were reported in the isolated dog heart (Andrus and Carter, 1924). In any case an important similarity between experimental acidosis leading to cardiac arrest in the bull calf and clinical cardiac arrest in man has been demonstrated. The clinical importance of the observation is discussed later.

Experimental Electrocardiographic Results. The present results illustrate (Fig. 2) the pattern of electrocardiographic changes which commonly occur during acidosis in artificially ventilated bull calves. There was great variation in individual tolerance to acidosis (Table II). Six animals showed a characteristic sequence in the presence of extreme acidosis: sinus tachycardia, electrical

alternans, two-to-one heart block, complete heart block, and asystole, with reversal of the sequence and restoration of cardiac function after the administration of bicarbonate. Atypical results, in which the heart beat could not be restored, occurred in four animals. Scrutiny of the data (Table II) suggested possible causes for the atypical responses (Fig. 3). In two animals (No. 2 and 10) with bradycardia the broad QRS complexes (Fig. 3B, C) were consistent with the hyperkalæmia present (Table II), and in the third animal (No. 6) the electrocardiographic changes (Fig. 3F) in the presence of an unrecordable blood pressure were consistent with circulatory arrest due to anoxia. In the fourth animal (No. 5) inconspicuous electrocardiographic changes (Fig. 3E) with early bradycardia and hypotension were followed by persistent ventricular fibrillation. It is perhaps significant that a large retroperitoneal hæmatoma, apparently caused during cannulation of the vessels, was found at necropsy. Hypoxia in addition to oligæmia may have been present in this animal also. It is probable that pulmonary œdema was an important contributory factor in some of the atypical results.

Comparison of Arrhythmias in Man and Calf. (i) *Heart Block.* In the intact bull calf the characteristic sequence of events seen in the electrocardiogram during extreme acidosis includes two-to-one heart block, complete heart block, and ventricular asystole and, in the restoration of sinus rhythm following infusion of bicarbonate solution, reversal of the sequence. A similar sequence may occur in clinical practice. Heart block was seen in all three clinical cases. In Case 3 an electrocardiogram (Fig. 1) 20 minutes after cardiac arrest, recorded during an intermission of external cardiac massage, showed complete heart block quickly changing to ventricular asystole (Fig. 1A). With re-inflation complete heart block recurred (Fig. 1B) and with more efficient intubation and vigorous inflation of the lungs this changed to two-to-one heart block which was maintained (Fig. 1C). Prompt improvement in atrio-ventricular conduction with restoration of sinus rhythm followed the administration of bicarbonate (Fig. 1D-F). When inflation was stopped, heart block (Fig. 1J) and ventricular asystole (Fig. 1K) recurred, but after external cardiac massage idio-ventricular rhythm was again present (Fig. 1M-O). These later changes were doubtless due to superadded hypercapnia or hypoxia or both.

(ii) *Electrical Alternans.* This phenomenon has frequently been seen in animal studies, but Colvin (1958), who reported its occurrence in a patient, found only 64 recorded clinical cases. It is of interest that this arrhythmia was present in one patient (Case 3, Fig. 1D) and was seen in almost all the experimental animals (Table II). Its pathogenesis remains uncertain but it is accepted that electrical alternans occurs in the absence of pulsus alternans.

(iii) *Ventricular Fibrillation.* Ventricular fibrillation was encountered in only one of the experimental animals and in two of the clinical cases. Spontaneous cessation of ventricular fibrillation was observed in Case 3 (Fig. 1K, L), and a similar occurrence has been recorded (Wetherill and Nixon, 1962) in a child treated by external massage. Usually, however, defibrillation would have to be secured by electro-shock.

Clinical Management. The importance of treating acidosis after cardiac arrest has been emphasized (Lancet, 1962) after the coincidental correction of arrhythmias was reported both in clinical practice (Stewart *et al.*, 1962) and in experimental animals (Ledingham and Norman, 1962). The present clinical cases illustrate some aspects of management. In Case 1, treated before the importance of correcting acidosis was recognized, a weak heart beat was restored by internal cardiac massage but later there was deterioration, at first with four-to-one heart block and subsequently with ventricular asystole. It seems probable in retrospect that the deterioration was not unrelated to acidosis. In Case 2 there was heart block which improved with internal massage. A blood sample after this episode was acidotic but the patient died in peripheral circulatory failure before the result was known. It has since been emphasized (Stewart, 1964) that the initial treatment of cardiac arrest should be undertaken on clinical assessment without waiting for the results of acid-base measurements. In Case 3, when severe acidosis was treated with intravenous sodium bicarbonate solution, the heart beat was restored, and the electrocardiographic sequence during correction of this acidosis (Fig. 1) was closely similar to that seen experimentally in the calf (Fig. 2).

Acidosis of mixed respiratory and metabolic type results from circulatory arrest which is not immediately treated. After cardiac massage and correction of respiratory acidosis, by inflation of the lungs with oxygen, the electrocardiogram may show heart block or asystole. The use of isoprenaline is advocated for heart block (Sodeman, 1958), and cardiac stimulants are advocated for asystole (Milstein, 1963) but in the first instance no time should be wasted with these. After cardiac arrest in a previously healthy heart such arrhythmias are most probably due to acidosis. 50 mEq of bicarbonate, a specific and rapidly acting remedy, should be given intravenously immediately and thereafter blood samples should be obtained for acid-base measurements which will indicate the need, if any, for further bicarbonate therapy. The clinical management of acidosis after cardiac arrest has been discussed by Stewart (1964).

SUMMARY

A characteristic sequence of electrocardiographic changes: sinus tachycardia, electrical alternans, two-to-one heart block, complete heart block, and ventricular asystole occurs in extreme metabolic acidosis induced experimentally in the bull calf. There is reversal of the sequence with correction of the acidosis. A similar sequence is seen during correction of acidosis in clinical practice. The importance of these observations in relation to the management of cardiac arrest in clinical practice is discussed.

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